

Mortality Impacts of Medicaid Expansion: Revisiting Sommers, et al. 2012

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ABSTRACT:

Background

Medicaid 1115 demonstration waivers allowed for states to expand benefits, largely to childless adults in poverty. Sommers, et al (2012) examined the mortality effects of these expansions using difference-in-differences methods.

Methods

We examine the same expansion states, and added two states with later expansions, but create a synthetic control group with a linear combination of county mortality rates in nearby states.

Results

Our methods showed a decline of 51 deaths per 100,000 population as a result of the Medicaid expansions.

Discussion

Our results support the findings of Sommers, et al, despite the difference in methodology used for identification.

BACKGROUND

The broad Medicaid expansion envisioned by the Affordable Care Act (ACA) in 2014 has sparked debate across numerous states on the benefits and costs of implementing such a policy. Despite the promise of significant federal funding to support the opportunity of expanding coverage to millions of citizens, opponents of expansion still question whether or not Medicaid expansion actually improves state health care systems, both in terms of cost reduction and gains in health outcomes.

Despite a number of analyses on the effect of Medicaid on health, little consensus has been reached. Studies of previous Medicaid expansions were focused on children and pregnant women, and yielded differing results as to whether expansion resulted in better health outcomes or not [1-4]. More recent studies have shown little to no effect of expanded insurance on mortality [5-7], and some studies have even found a negative correlation between Medicaid coverage and outcomes [8, 9]. However, many of these studies suffer from methodological limitations; the absence of a strong counterfactual comparison limits the usefulness of these results in terms of establishing the causal effect of Medicaid expansion on health outcomes.

More recently, Sommers et al [10] used a difference-in-difference approach to compare states that implemented significant Medicaid expansion in the early 2000's with similar neighboring states that did not. This analysis concluded that Medicaid expansion was associated with reduced mortality and improved insurance coverage, access to care, and self-reported health. By using data from multiple sources, and establishing significant changes in variables along the expected causal pathway of mortality reduction (i.e. health insurance coverage, delaying of medical care due to cost, etc.) this analysis makes a compelling claim as to the likely effects of Medicaid expansion.

However, the appropriateness of the counterfactual comparisons used and overwhelming influence of New York data in the original analysis raise questions around the generalizability of these findings. The authors acknowledge that the overall findings are driven by the large population in New York, and thus the appropriateness of using Pennsylvania as a control for this state comes into question. The unique diversity and size of New York City in particular would seem to be very difficult factors to find an appropriate control for, especially since entire states were used as counterfactual comparisons.

In this paper we present a re-analysis of the Sommers et al paper, first replicating the original results (see Appendix B), and then extending the analytic timeframe of pre-post expansion years in order to explore longer-term effects of Medicaid expansion. In order to address the limitations of state-level counterfactuals we demonstrate the construction and use of county-based synthetic counterfactuals for each demographic subgroup of interest.

METHODS

In addition to the three expansion states included in the original analysis (Arizona, Maine, New York), we included two other states that experienced Medicaid expansions in the years shortly following the original analysis (Iowa and Maryland). To provide control data we also included neighboring states, with the exception of Massachusetts due to the comprehensive health policy reforms that occurred during this period.

For each state we obtained county-level mortality data by age (20-24, 25-34, 35-44, 45-54, 55-64), sex, and race (White, Black, Other). Younger ages and adults 65+ were excluded due to Medicaid policies for children, and Medicare for older adults. Hispanic origin is not available for CDC mortality data prior to 1999, and so was not considered in this analysis.

From the Compressed Mortality File we obtained data for the states of interest for 1979-2014, yielding 1,350,766 subgroup-specific observations. After restricting the years to 1990-2014 and removing county age-sex-race subgroups for which at least one year of population data was suppressed due to small sample size, our final dataset consisted of 21,565 subgroup-counties, each of which had 25 years of data, yielding 539,125 observations in total.

For each age-sex-race subgroup in an expansion state, we constructed a synthetic counterfactual by taking a linear combination of demographic-specific county data in neighboring states. Each potential control county data point was given a "weight" such that the overall distance-squared between the pre-treatment

mortality in the treatment and control counties was minimized, subject to a penalty parameter on the L-1 norm. The equation was as follows:

$$Y_{a,s,r,y,c} = \alpha_{a,s,r} + \sum_{k=1}^K \beta_{a,s,r,k} X_{a,s,r,k,y} + \epsilon_{a,s,r,y,c},$$

$$\hat{\beta}_k = \min_{\beta} E \omega_{a,s,r,y,c} \left(Y_{a,s,r,y} - \alpha - \sum_{k=1}^K \beta_k X_{a,s,r,y} \right)^2 - \lambda \sum_{k=1}^K |\beta_k|$$

where (a,s,r) denote an age, sex, race subgroup, for year y in the pre-treatment period, for treatment county c . Coefficients β_k denote the contribution of each control county k in predicting the average treatment county subgroup a,s,r in year y . The coefficient was estimated using weighted least angle regression using the weight $\omega_{a,s,r,y,c}$. The penalty parameter (λ) was tuned to the minimum out-of-sample mean square error using 5-fold cross-validation. After the coefficients were estimated, the synthetic control estimate $\hat{Y}_{a,s,r,y}$ was calculated for the full file: both pre- and post-treatment.

Having created the synthetic control group, we then estimated difference-in-difference (DD) models for the pooled sample of Arizona, Maine, and New York and each state individually. Because the synthetic controls were matched on pre-treatment mortality for population sub-groups, there was no need to include additional covariates in the model. Both the treatment and synthetic control groups use the same treatment county subgroup population totals as weights to account for the differential contribution of each county subgroup to the state estimate.

In addition to the DD models, we estimated interrupted time-series models, specified as linear trends in the pre- and post-treatment period. The model is as follows:

$$Y = \beta_0 + \beta_1 t + \beta_2 POST \cdot t + \beta_3 TREAT \cdot POST \cdot t + \epsilon$$

where Y combines the actual (treatment) and synthetic (control) mortality rate for county subgroups. The year variable is transformed into a relative time variable (t) by subtracting the year of Medicaid expansion such that negative values indicate the pre-treatment period and positive ones indicate the post-treatment period. In this model, β_0 is the mortality rate in the year of implementation and β_1 is the linear change in mortality rate during the pre-treatment period. $(\beta_1 + \beta_2)$ is the change in mortality rate in the post-period for the synthetic control group while $(\beta_1 + \beta_2 + \beta_3)$ is the change in mortality rate in the post-period for the treatment group. Thus, β_3 is the change in linear trend due to the Medicaid expansion.

All analyses were performed in R. Replication datasets and code are available (see Appendix C).

RESULTS

Figure 1A presents our replication results of the original analytic timeframe used in the Sommers et al analysis (see Appendix B for further replication results). Figure 1B presents the expanded analytic period of 1990-2014 used in this analysis. Mortality rates (expressed in death per 100,000 individuals) in the

expansion states drop from 392 in 1994 to 315 in 1999. During this same period, mortality in control states dropped from 362 in 1994 to 340 in 1999. Looking at this longer timeframe, we find that the treatment states experienced large decreases in mortality prior to Medicaid expansion that was not detected in the original analytic period.

Figure 1. Crude mortality rate (per 100,000) for treatment and control states: Original Analytic Period (1997-2005) and Expanded Analytic Period (1990-2014)

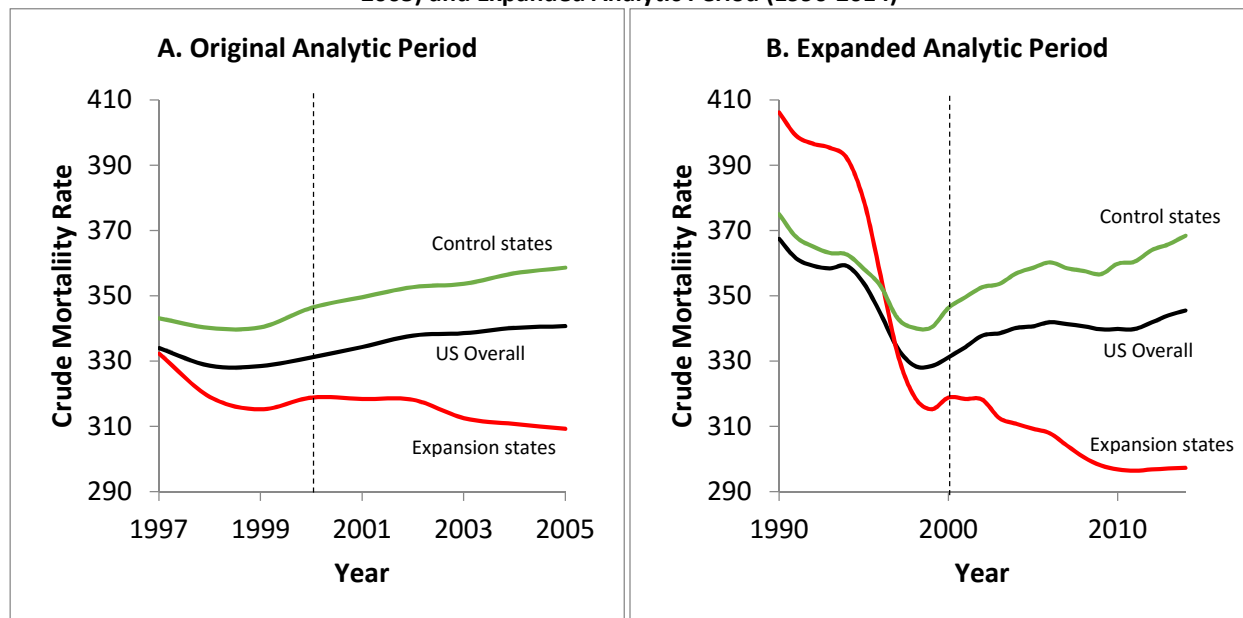


Figure 1.A shows the original analytic timeframe. The dashed line indicates the Medicaid expansion period. Figure 1.B shows a significant shift in mortality between the control and treatment groups, with the treatment group experiencing large decreases in mortality prior to the expansion period. (Data were smoothed with a 3-year moving average for ease of visualization.)

We further investigated the reliance of the previous analyses' results on mortality trends in individual states (see Appendix A), and New York in particular (see Figure 2). Panel A in figure 2 presents external-cause mortality (deaths caused by external or environmental factors such as accidents or crime) in New York from 1990-2014 split by race and sex subgroups. While most race and subgroup combinations remained stable through this time period, external cause mortality (reported in deaths per 100,000 individuals) in African-American males declined from 177 in 1992 to 92 in 1999. Panel B shows HIV/AIDS-specific mortality in New York from 1990-2014. With the introduction of combination therapy in 1996, large decreases in HIV/AIDS-specific mortality were observed. HIV/AIDS mortality in African-American males dropped from 251 deaths per 100,000 individuals in 1995 to 80 deaths in 1999, and there was also a significant decrease for African-American females and White males during this period.

Figure 3 presents the results of our pooled analysis using synthetic controls developed in place of the states chosen in Sommers et al. Table 1 shows the estimates from the difference-in-difference models utilizing the synthetic mix of states as the control group, both for the pooled analysis as well as the individual states of Arizona, Maine, and New York. The pooled analysis shows a significant decrease in mortality for the post-treatment period in the treatment group, with a decrease of 51 deaths per 100,000

($p < 0.001$). Within the individual states, Maine and New York both showed significant changes in post-treatment differences between treatment and synthetic controls, while Arizona did not.

Figure 2. Cause-specific mortality rates (per 100,000) by race and sex in New York, 1990-2014

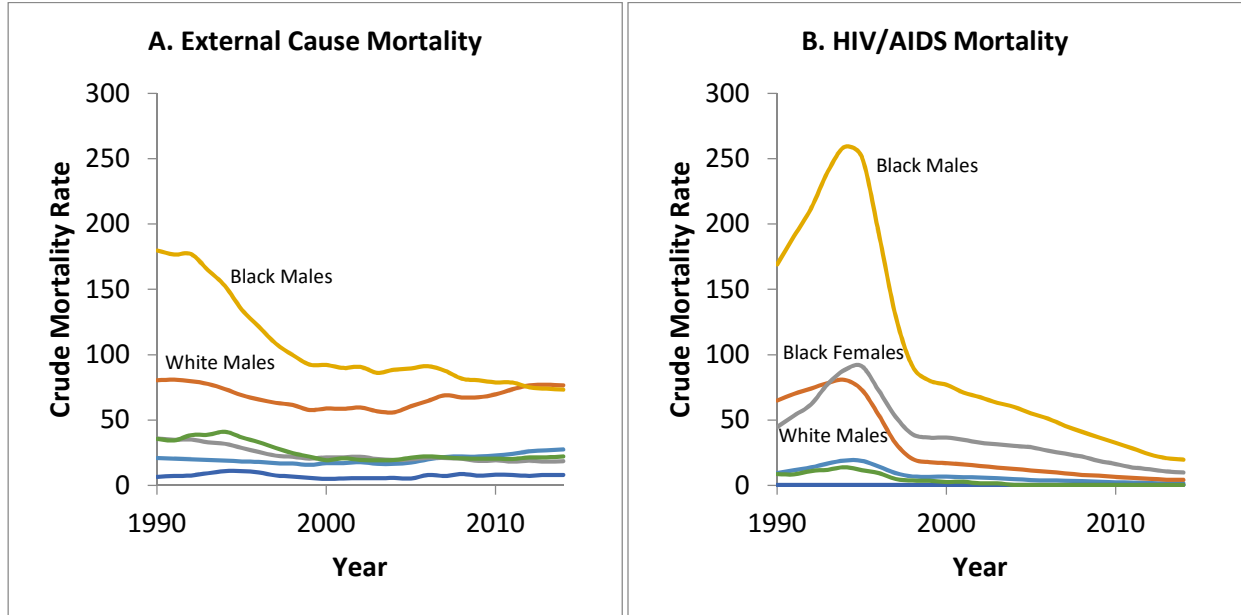


Figure 2 highlights cause-specific mortality rates in New York. Among black males, external cause mortality dropped by 85 deaths per 100,000 between 1992-1999, and HIV-specific mortality dropped sharply from 251 in 1995 to 80 in 1999.

Figure 3. Crude mortality rate (per 100,000) for expansion states and synthetic controls

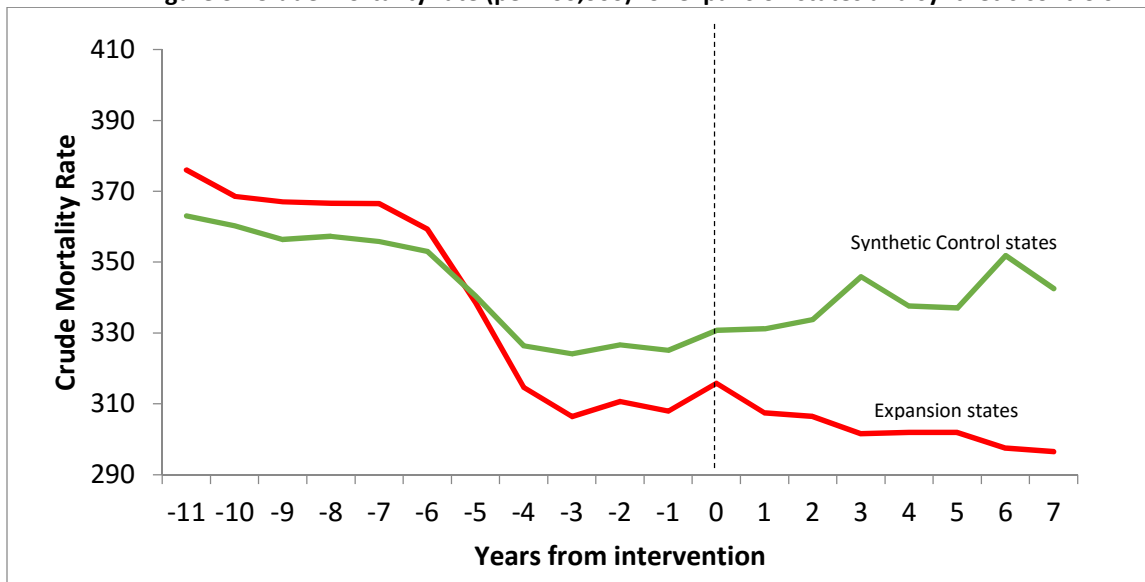


Figure 3 presents the pooled mortality trends in expansion states compared to the controls, which consist of a synthetic mix of neighboring states matching on demographic-specific pre-treatment mortality.

Table 1. Estimate of Difference-in-Difference Models with a Synthetic Control Group

	Pooled	AZ	ME	NY
Intercept	353.1 *** (2.2)	329.1 *** (5.3)	300.0 *** (6.6)	362.2 *** (2.7)
Treatment Group (<i>treat</i>)	0.0 (3.2)	0.0 (7.5)	-0.5 (9.4)	0.0 (3.8)
Post-treatment Period (<i>post</i>)	-3.5 (3.2)	19.3 ** (7.1)	105.0 *** (9.4)	-16.1 *** (3.8)
DD estimate (<i>treat x post</i>)	-51.0 *** (4.5)	-17.4 (10.1)	-77.5 *** (13.3)	-59.2 *** (5.4)

Standard Errors in parentheses. *** p<0.001, ** p<0.01, * p<0.05

Table 2. Estimate of Interrupted Time Series Models with a Synthetic Control Group

	Pooled	AZ	ME	NY
Intercept	315.0 *** (2.2)	328.8 *** (5.0)	319.1 *** (6.6)	311.5 *** (2.7)
Time	-7.3 *** (0.4)	-0.3 (0.9)	2.5 * (1.1)	-9.5 *** (0.5)
Time x Post	12.5 *** (0.7)	3.5 * (1.6)	9.6 *** (2.1)	14.6 *** (0.9)
Time x Post x Treatment	-7.7 *** (0.5)	-3.1 ** (1.0)	-10.5 *** (1.4)	-9.0 *** (0.6)

Standard Errors in parentheses. *** p<0.001, ** p<0.01, * p<0.05

Table 3. Extension of models to new states (Iowa & Maryland) with Synthetic Controls

	Iowa		Maryland	
	DD	ITS	DD	ITS
Intercept	236.9 *** (1.6)	243.9 *** (2.7)	351.2 *** (4.2)	348.6 *** (4.6)
Post	-1.7 (2.6)		18.9 ** (6.8)	
Treatment	-2.1 (2.3)		-10.9 (6.0)	
Post x Treatment	-37.1 *** (3.7)		-41.6 *** (9.6)	
Time		1.4 ** (0.4)		0.6 (0.8)
Time x Post		6.7 *** (1.2)		3.9 * (2.0)
Time x Post x Treatment		-4.0 *** (1.0)		-11.0 *** (1.7)

Standard Errors in parentheses. *** p<0.001, ** p<0.01, * p<0.05

Table 2 shows estimates from the interrupted time series model using the synthetic states as a control. The individual state analyses generally followed the pooled analysis, which estimated a 7.7 deaths per 100,000 decrease per year in the treatment states post-intervention.

The additions of Iowa and Maryland into the analysis are shown in table 3. The post-treatment reduction in mortality is 37.1 deaths per 100,000 in Iowa and 41.6 deaths per 100,000 in Maryland. The interrupted time series analysis shows significant linear changes in mortality reduction in pre- and post- treatment

control group and post-treatment treatment group in Iowa, while only post-treatment control and treatment groups had significant linear changes in mortality reduction from baseline (year of intervention).

DISCUSSION

This study investigates the parallel trends assumption in Sommers et al between treatment states that underwent Medicaid expansion compared to control states which did not. We explored the state-level trends and found that trends in New York significantly differed from other studied states, with large reductions in mortality occurring in the mid 1990's.

In further investigating the mortality reduction within New York, we find that much of the sharp decline is explained by reductions in external cause mortality in African-American males, possibly due to studied decreases in violent crime around this time. In addition, with the introduction of combination therapy for HIV in 1996, we see a sharp reduction in HIV-specific mortality. Together, these two phenomena explain a large portion of the overall mortality trends in New York.

In their limitations, Sommers et al mention the size of New York as the driving force in the overall results, raising concerns about generalizability. In addition to issues of external generalizability, however, we find that there may also be a significant threat to internal validity in the use of Pennsylvania as a comparator state. Because of the significant changes in subgroup mortality shortly prior to Medicaid expansion in New York, it is difficult to make comparisons at the state-level on mortality trends. To account for this, we conducted the analysis with synthetic controls, using a mix of county-level data in states neighboring the treatment states. Because the synthetic estimates were only trained on pre-treatment period data, the predictions in the post-period reflect the predicted values for treatment states in the absence of the Medicaid expansion.

In the end, our results support the overall findings of Sommers et al that Medicaid expansions have resulted in decreased mortality rates, and we show that these results are robust to changes in methodology. In fact, we find stronger mortality reductions (51 deaths per 100,000 compared to 20 in Sommers et al) at both the pooled and state level. Furthermore, we studied two states, Iowa and Maryland, that expanded Medicaid after the study period of Sommers et al and find that both states had similar reductions in mortality following Medicaid expansion.

However, there are a number of limitations to our analyses. Due to the nature of the data we were not able to control for individual-level characteristics. Our method of matching county-level data within subgroups accounts for differential mortality across the subgroups we defined, but there may be other factors affecting mortality rates over time that were not accounted for. We also assume that the synthetic mortality trends trained on pre-treatment data continue in the post-treatment period. By matching within demographic groups, we control for any changes due to demographic composition at the state-level. In addition, by using a coarsened exact matching approach within these subgroups, we preserve the dimensionality of mortality differences. This has been shown to be a superior method to lower dimensional matching methods such as propensity scores as used recently by Sommers et al[11] in

analyzing health reform in Massachusetts. Finally, the inherent nonrandomized nature of Medicaid expansion means care must be taken when comparing treatment and control states.

We also addressed two major limitations related to generalizability by analyzing Iowa and Maryland separately. First, we demonstrated that the results are robust to possible period effects since Iowa and Maryland expanded Medicaid at a later time. Secondly, we are able to ensure that a large, unique state such as New York was not driving the results of the analysis, thus demonstrating the generalizability of the results to other time periods and states.

In conclusion, we have demonstrated a novel approach for counterfactual comparisons to study the effect of Medicaid expansion on health outcomes. By creating synthetic controls that are similar in terms of salient pre-treatment characteristics, we were able to generate post-treatment results that are less sensitive to limitations in previous analyses. Utilizing this approach may allow us to further investigate effects of population-level policy changes on health outcomes.

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